

AMENDMENTS TO THE CLAIMS:

1-15. (Canceled)

16. (Currently Amended) A method for treating at least one autoimmune condition promoted by an increase in IFN- γ and/or TNF- α levels in a human subject, said method comprising administering to said subject a therapeutically effective amount of at least one antagonist that binds with a the 40 kD subunit of IL-12, wherein said antagonist is chosen from at least one antibody immunoreactive with the 40 kD subunit and at least one antibody fragment immunoreactive with the 40 kD subunit.

17. (Canceled)

18. (Previously Presented) The method of claim 16, wherein the antibody is a monoclonal antibody.

19. (Previously Presented) The method of claim 16, wherein the antibody is a polyclonal antibody.

20. (Previously Presented) The method of claim 16, wherein the 40 kD subunit is disulfide-bonded to the 35 kD subunit of IL-12.

21. (Previously Presented) The method of treating at least one autoimmune condition of claim 16, wherein the antagonist either

(a) blocks the formation of a heterodimer containing the 40 kD subunit; or

(b) allows the formation of a heterodimer containing the 40 kD subunit, but blocks the activity of said heterodimer.

22. (Previously Presented) The method of claim 21, wherein the autoimmune condition is chosen from multiple sclerosis, systemic lupus erythematosus, rheumatoid

arthritis, autoimmune pulmonary inflammation, Guillain-Barre syndrome, autoimmune thyroiditis, insulin dependent diabetes mellitus, and autoimmune inflammatory eye disease.

23. (Previously Presented) The method of claim 21, wherein the autoimmune condition is insulin dependent diabetes mellitus.

24. (Previously Presented) The method of claim 21, wherein the autoimmune condition is systemic lupus erythematosus.

25. (Currently Amended) A method for treating at least one autoimmune condition promoted by an increase in IFN- γ and/or TNF- α levels in a human subject, said method comprising administering to said subject a therapeutically effective amount of at least one antagonist that binds with a the 35 kD subunit of IL-12, wherein said antagonist is chosen from at least one antibody immunoreactive with the 35 kD subunit and at least one antibody fragment immunoreactive with the 35 kD subunit.

26. (Canceled)

27. (Previously Presented) The method of claim 25, wherein the antibody is a monoclonal antibody.

28. (Previously Presented) The method of claim 25, wherein the antibody is a polyclonal antibody.

29. (Previously Presented) The method of claim 25, wherein the 35 kD subunit is disulfide-bonded to the 40 kD subunit of IL-12.

30. (Previously Presented) The method for treating at least one autoimmune condition of claim 25, wherein the antagonist either

(a) blocks the formation of a heterodimer containing the 35 kD subunit; or

(b) allows the formation of a heterodimer containing the 35 kD subunit, but blocks the activity of said heterodimer.

31. (Previously Presented) The method of claim 30, wherein the autoimmune condition is chosen from multiple sclerosis, systemic lupus erythematosus, rheumatoid arthritis, autoimmune pulmonary inflammation, Guillain-Barre syndrome, autoimmune thyroiditis, insulin dependent diabetes mellitus, and autoimmune inflammatory eye disease.

32. (Previously Presented) The method of claim 30, wherein the autoimmune condition is insulin dependent diabetes mellitus.

33. (Previously Presented) The method of claim 30, wherein the autoimmune condition is systemic lupus erythematosus.